ABSTRACTS OF CARDIOLOGY

Pulmonary Atresia and the Collateral Circulation to the Lungs. K. D. ALLANBY, W. D. BRINTON, M. CAMPBELL, and F. GARDNER. *Guy's Hosp. Rep.*, **99**, 110–152, 1950. 22 figs., 22 refs.

The clinical and anatomical features in 6 cases of pulmonary atresia are described. In one patient, aged 15, with a closed ventricular septum, the pulmonary circulation was maintained by a widely patent ductus communicating with a grossly dilated pulmonary artery; the conclusion was reached that the pulmonary blood supply in this instance was better than in many cases of tetralogy. The other 5 cases were of pulmonary atresia with high ventricular septal defect and over-riding aorta. Atresia was usually at, or near, the pulmonary valve, the pulmonary artery being small, but patent distal to the atresia. Abnormally large bronchial arteries were found in every case and in some were joined directly with branches of the pulmonary artery. The development and classification of these vessels is discussed. The authors feel that this group of cases with pulmonary atresia should be separated from their conventional classification with the tetralogy and be considered as a distinct entity; only cases with pulmonary stenosis should be defined as the tetralogy of Fallot. On the clinical side the authors make the point that in pulmonary atresia a systolic murmur is generally absent and the second sound in the pulmonary area, is louder than in the tetralogy. [The article is exceptionally well illustrated and repays close study.] *James W. Brown*

Pheochromocytoma and Essential Hypertensive Vascular Disease. M. GOLDENBERG, H. ARANOW, A. A. SMITH, and M. FABER. *Arch. intern. Med.*, **86**, 823–836, Dec., 1950.

This is a comprehensive paper based on the findings in 22 cases of pheochromocytoma, of which the clinical histories are given. These tumours contain adrenaline and noradrenaline in varying proportions. Continuous intravenous infusion of the former raises the systolic blood pressure, causes hyperglycæmia, augments the basal metabolic rate (B.M.R.), and produces anxiety and palpitations, whereas noradrenaline raises both the systolic and diastolic pressures, has little effect on the B.M.R., and produces the picture of classical essential hypertension, which may be mimicked in cases of pheochromocytoma.

It was found that in 7 of the 12 cases in which the pheochromocytoma was removed surgically the hypertension continued after the operation but, strangely enough, the symptoms disappeared. It would appear that although the primary stimulus had ceased, the hypertension was irreversible [compare the results of nephrectomy for hypertension]. Chemical analysis of 15 of the tumours was undertaken and it was found that where the tumour contained a large amount of adrenaline, the clinical picture was one of tachycardia, high B.M.R., and hyperglycæmia, whereas small tumours, which contained only noradrenaline, produced the pure picture of essential hypertension. The former group usually showed a strong positive reaction to the piperoxan hydrochloride test. *Paul B. Woolley*

The Ventricular Electrokymogram. L. C. AKMAN, A. J. MILLER, E. N. SIBLER, J. A. SCHACK, and L. N. KATZ. *Circulation*, **2**, 890–899, 1950. 8 figs., 17 refs.

The apparatus used in this study consisted of a Sanborn electrokymograph and a Sanborn stethocardiette for recording of the heart sounds, which were used for time reference. Over 200 left ventricular electrokymographic tracings were obtained in 32 young healthy adults. On the average six different sites over the left ventricle were explored in each case; the curves obtained were analysed and compared. The authors present in two tables the relevant measurements and the range of variation as between one tracing and another in the same individual. The contour variations are described in detail and illustrative examples reproduced.

It is concluded that variations in contour and time sequence of events in the normal electrokymogram are considerable. These variations are caused not so much by changes in cardiac volume as by positional movements of either the whole or parts of the heart. Furthermore, it was found that there was no consistent moment-to-moment time relationship between electrokymograms obtained at various sites on the left ventricular border and the heart sounds.

It is pointed out that certain curve patterns hitherto described as abnormal and characteristic of myocardial infarction and constrictive pericarditis might occur as a normal variant in electrokymograms from healthy hearts. *A. I. Suchett-Kaye*

Intravenous Use of Quinidine, with Particular Reference to Ventricular Tachycardia. A. H. CLAGETT. *Amer. J. med. Sci.*, **220**, 381–388, 1950.

Although quinidine has been given by mouth in the treatment of ventricular tachycardia since 1922 there are comparatively few references in the literature to its intravenous use. Out of 97 recorded cases, of which 62 were of paroxysmal ventricular tachycardia, in 42 the rhythm reverted to normal and only in 11 cases did reactions occur, 4 of which (all in the same series) were fatal.

In the authors opinion, the intravenous injection of

quinidine is justified when rapid action is needed or when the patient is unable to take it by mouth owing to gastro-intestinal upset. He uses ampoules of quinidine lactate containing 0·65 g. in 10 ml. and adds one of these to 50 ml. of 5% glucose, giving the solution intravenously at approximately 2 ml. a minute.

The treatment of 13 cases is described in detail.

M. H. Pappworth

Text Book of Radiological Differential Diagnosis. Vol. I. Diseases of the Thoracic Organs. (Lehrbuch der Roentgenologischen Differential Diagnostik Band. I Erkrankungen der Brustogane). WERNER TESCHENDORF, Cologne. Gearg Thieme: Stuttgart, 1950.

This volume gives a carefully thought out description of the radiology of the chest including the œsophagus. The first half of the book deals with diseases of the lung and pleura.

The author gives an interesting account of the differential diagnosis of aortic aneurysm, pointing out that whereas the kymograph accurately records the movements present, these may be absent in the aneurysmal sac owing to clot, but present in some cysts owing to transmitted pulsations.

The section on the heart, which comprises about one-fifth of the book, deals first with methods of measurement of heart size using orthodiagrams and teleradiography. The uses of kymography are thoroughly discussed, one of the advantages of which is that the amplitude and form of the waves can be used as a method of assessment of heat function.

There is a short section on congenital abnormalities of the heart, but angiocardiography is not discussed except to point out its potential dangers. The changes seen in disturbances of heart rhythm in the kymograph are contrasted with their much more exact demonstration by electrocardiography. Some interesting illustrations of kymographs are given of patients with heart block, especially complete heart block when the slow rounded ventricular waves with lateral plateau formation are well seen. In some cases the more rapid auricular contractions are discernible.

Diseases and abnormalities oft he œsophagus are discussed, and the last part of the book deals with the diaphragm.

This book is beautifully produced with clear print and marginal summaries of each paragraph. The 865 illustrations are all very good and show the points under discussion clearly. It is a compendium of information and is strongly recommended as a reference book and also as a textbook for students of chest radiology. There is an excellent bibliography.

F. G. Wood

Left Ventricular Pressures in Patients with Aortic Insufficiency Studied by Intracardiac Catheterization. H. A. ZIMMERMAN. *J. clin. Invest.*, **29**, 1601–1603, Dec., 1950.

The pressure levels within the left ventricular cavity have been studied in 10 human subjects with aortic insufficiency by catheterization of the left ventricle via the radial artery. The 3 patients who exhibited no clinical evidence of congestive failure had normal left ventricular diastolic pressures. The 7 patients in obvious congestive failure demonstrated significant increases in diastolic pressure averaging 25 mm. of mercury.—[Author's summary.]

Pulmonary Hypertension in Mitral Stenosis. R. I. S. BAYLISS, M. J. ETHERIDGE, and A. L. HYMAN. *Lancet*, **2**, 889–894, Dec. 30, 1950.

In 22 patients with mitral stenosis the right ventricular and/or pulmonary artery pressures have been correlated with clinical, radiological, and electrocardiographic findings. Pulmonary hypertension at rest was found in 17 patients. In general, those with the most severe symptoms had the highest pressures, but the duration of symptoms was not related to the degree of pulmonary hypertension.

Patients with severe pulmonary hypertension without peripheral venous congestion presented a recognizable syndrome, characterized by predominant pulmonary symptoms and electrocardiographic evidence of right ventricular hypertrophy. Patients with lesser degrees of pulmonary hypertension did not present characteristic features, but right ventricular enlargement on fluoroscopy was a constant finding. The degree of pulmonary hypertension, however, can be determined only by direct measurement. Right ventricular enlargement was found in all patients with pulmonary hypertension. There was no relation between the size of the left atrium and the degree of pulmonary hypertension.

On exercise a striking rise in pulmonary artery pressure occurred without a significant increase in cardiac output. The pulmonary hypertension at rest and on exercise is due to stenosis of the mitral valve and to increased pulmonary arteriolar resistance. The relative importance of these two factors is discussed.—[Authors' summary.]

Effects of Exercise on Circulatory Dynamics in Mitral Stenosis. III. R. GORLIN, C. G. SAWYER, F. W. HAYNES, W. T. GOODALE, and L. DEXTER. *Amer. Heart J.*, **41**, 192–203, Feb., 1951.

The authors report 9 observations on 8 cases of mitral stenosis with minimal disease of other valves. All but the most severe clinical grades of stenosis were represented, only patients thought to be unable to exercise safely being excluded. Despite this selection, 3 patients developed pulmonary œdema during the investigation.

Pulmonary "capillary" pressure was recorded at rest, and then during exercise performed (while recumbent) on a bicycle ergometer. Exercise increased the oxygen consumption to 1·5 to 3 times the resting level. In 8 observations there was no change in arterial oxygen saturation, and in the 5 patients with a low fixed cardiac output there was a proportional increase in arteriovenous oxygen difference. The mean resting pulmonary "capillary" pressure was raised in all cases and further increased with exercise. The pulmonary arterial pressure rose with the pulmonary "capillary" pressure, and the "PA–PC" gradient was unchanged, or rose only 1 to 15 mm. Hg. The calculated pulmonary arteriolar

resistance was unchanged by exercise in 2 cases, and rose in 2 cases.

The authors suggest that cardiac output becomes fixed in mitral stenosis because of: (1) the very high pulmonary arteriolar resistance, (2) the limitation of the patient's effort by pulmonary congestion, and (3) the decrease in stroke volume of the right ventricle resulting from the fixed output and the tachycardia on exercise. Three patients were othopnœic at rest and had high resting pulmonary "capillary" pressures (26 to 34 mm. Hg). On exercise these patients developed pulmonary œdema, with a rise in pulmonary capillary pressure to 35 to 47 mm. Hg. In other cases, in which the resting value was lower, no œdema developed despite a rise above 35 mm. Hg on exercise. *D. Verel*

Mural Thrombosis and Arterial Embolism in Mitral Stenosis. A Clinico-pathologic Study of Fifty-one Cases. R. A. JORDAN, C. H. SCHEIFLEY, and J. E. EDWARDS. *Circulation*, **3**, 363–367, March, 1951.

Out of 11,536 necropsies performed at the Mayo Clinic during the 20-year period 1929 to 1948 there were 51 cases of mitral stenosis with intracardiac mural thrombi. Of the 42 cases in which mural thrombi were present in the left side of the heart (including 15 in which the right side was also involved) they were restricted to the auricular appendage in only 20. It would seem, therefore, that surgical resection of the left auricular appendix in such patients would offer no more than a 50% chance of eliminating the source of systemic arterial emboli. On the other hand, right atrial thrombi were restricted to the auricular appendix in 20 out of 24 cases, but embolism from thrombi in peripheral veins would still have to be reckoned with when considering surgical resection or obliteration of this appendix for repeated pulmonary infarction. *T. Semple*

The Natural History of the Electrocardiogram in Mitral Stenosis. [In English]. H. RASMUSSEN and J. BOE. *Cardiologia*, **18**, 33–44, 1951.

Serial electrocardiograms (standard limb leads) were recorded in 50 patients with mitral stenosis over periods ranging from 1 to 18 years (average 6 years). The findings confirmed the presence in such patients of "characteristic, but not specific, electrocardiographic changes." In 18 cases they observed the development in lead I of a low R, a broad-based straddling R, notching of R, or a second small wave following the main R wave. In 11 cases right axis deviation developed, while in 7 a right ventricular retardation (type I) developed. In attempting to correlate the presence of these changes with the size of the heart, it was noted that of 17 patients in whom there was considerable increase in size of the heart, 15 had electrocardiographic changes, while of 18 with no increase in size of the heart, 12 had such changes. For 10 years or more no changes might be noted, then within a matter of months marked changes would occur. This is taken to be an argument in favour of the view that dilatation rather than hypertrophy of the right ventricle is the main factor responsible for the electrocardiographic changes. Six unipolar præcordial leads were used in the final records taken on all 50 patients and showed relatively few significant changes compared with the limb leads. When first observed, 21 of the patients had sinus rhythm, compared with only 9 at the final examination. The corresponding figures for persistent auricular fibrillation were 24 and 39.

William A. R. Thomson

Surgery of Stenotic Valvular Disease of the Heart. R. P. GLOVER, C. P. BAILEY, and T. J. E. O'NEILL. *J. Amer. med. Ass.*, **144**, 1049–1057, Nov. 25, 1950.

In this review of the development of intracardiac operations for stenotic valvular disease, the results so far obtained in Philadelphia are described.

Congenital pulmonary stenosis. Eight cases have been operated upon with no death. In cases of the valvular type of stenosis (4 cases) the Brock technique was used, but for the infundibular type of stenosis (4 cases) a segment of septal tissue is removed with a sharp bone rongeur through an incision in the myocardium extending vertically above and below the septal ridge and at right angles to it. The early results at least equal those obtained by shunt procedures.

Mitral stenosis. In cases suitable for operation there should be no evidence of rheumatic activity, and sinus rhythm should be normal. Uncontrolled cardiac failure or appreciable left ventricular enlargement is a contraindication. Commissurotomy has been performed on 50 patients with 8 deaths. On the criteria of clinical disappearance of presenting symptoms and the reduction of left auricular and over-all heart size, excellent results have been obtained in 20 cases. The remainder have been improved.

Aortic stenosis. The contracted aortic valve can be approached from below via the left ventricle or from above via the common carotid artery. A Brock type of operation was performed on one patient, but the resulting aortic regurgitation caused the death of the patient. In two further cases the valve was successfully enlarged by passing a special dilator through the valve opening from above via a cervical incision into the right common carotid artery. *R. Lambert Hurt*

Atrial Flutter. II. Methods of Treatment. G. R. HERRMANN and M. R. HEJTMANCIK. *Amer. Heart J.*, **41**, 182–191, Feb., 1951.

The authors review published accounts of the treatment of auricular flutter and analyse the results in 93 episodes occurring in 82 new cases. In 28 cases in which the flutter was of less than 72 hours' duration sinus rhythm returned without therapy in 8. In flutter lasting more than 72 hours digitalis folia was effective in 19 of 24 cases, 7 being left with persistent fibrillation. Digitalis glycosides were effective in 5 of 8 cases, one persisting in fibrillation. Quinidine was effective in 11 of 21 cases, and with digitalis in 4 of 8 cases. Intravenous procaine failed in 2 cases.

The authors conclude that digitalis folia is the drug of choice in flutter where there is serious heart disease. Quinidine orally is effective, but should be given only in the absence of contraindications. In refractory cases intravenous quinidine may be tried, with constant observation of blood pressure and pulse rate. *D. Verel*